POCKET HANDBOOK

. . . FOR THE RECOGNITION AND DIAGNOSIS OF CERTAIN

ANIMAL DISEASES EXOTIC TO MOST OF THE AMERICAS

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*PRESENT IN NORTH AMERICA

**PRESENT IN SOUTH AMERICA

***NOT ILLUSTRATED

PREFACE

This booklet is a "miniaturized" summary describing the key features of and diagnostic approaches to selected exotic contagious diseases which are hazardous to livestock and poultry in the Americas. It has been kept pocket size for handy reference.

The information has been "scaled down" to serve as a memory "jogger" - first aid - for consultation incident to an emergency call to investigate a suspected exotic animal disease. Major clinical features of the disease are included. In the appended color microfiche are photographs of typical and pathognomonic lesions. The microfiche may be read in a commercial reader, by means of any 10X magnifier, under a photographic enlarger, or a dissecting microscope. Copies of the material may be made with a photographic enlarger. To obtain the most benefit from the booklet, the reader should be somewhat familiar with large animal and poultry disease syndromes generally. It is expected that the reader will wish to augment this initial short review with study of appropriate literature in depth. Examples of such literature on each disease are listed at the end of each textual description.

A more comprehensive book that covers all of these diseases and others is the "gray book" entitled "Foreign Animal Diseases," published by the U.S. Animal Health Association, 1964. (This publication is currently being revised; a new edition is expected in 1975.)

1. RINDERPEST

1. Definition:

Rinderpest (RP) or cattle plague is an acute, highly contagious virus disease, primarily of cattle, secondarily of sheep, goats, and wild ruminants. Pigs of European and North American origin, when exposed to rinderpest, may develop an inapparent infection with a mild transient fever (although they may transmit virulent virus to cattle). The American javelina and indigenous swine of the Far East are highly susceptible.

2. Etiology:

Rinderpest virus strains are immunologically uniform, but they may vary in virulence. The virus is about 300 nm in size and immunologically related to measles and distemper viruses. It is destroyed by strong acids and alkalies.

3. Geographical Distribution:

The disease is enzootic in Asia and Africa but not in Europe or the Americas. The last epizootic in Europe was in Belgium in 1920.

4. Transmission:

Transmission is by contact with infected animals or indirectly with their secretions, excretions, and fomites. The virus appears in the blood and secretions before the appearance of signs. For this reason, the infection may be easily introduced inadvertently to slaughterhouses and stockyards. Animals that recover develop solid immunity and a high antibody titer; they are not known to be carriers.

5. Hosts:

Hosts are chiefly cattle, buffaloes, deer, camels, sheep, goats, and occasionally swine.

6. Clinical Signs:

The incubation period is ordinarily 3 to 10 days, although where the disease is enzootic it may be longer; the incubation period of the experimental disease may be as short as 40 hours.

The major clinical signs are high fever, nasal discharge, erosions of the buccal mucous membrances, constipation followed by diarrhea, dehydration, rough and soiled hair coat, and death in 7 to 12 days.

7. Gross Lesions:

Lesions include punched-out-like erosions on the inner surfaces of the lower lip, the gums, ventral surface of the tongue and the soft palate. Upon necropsy, the lymph nodes are edematous. Peyer's patches are acutely inflamed, eroded, severely hemorrhagic, and necrotic. The mucosa of the abomasum is hemorrhagic. There is often edema, hemorrhage, and erosions of the mucosa of the cecum, the cecocolic junction and the rectum. The mucosal surface of the last portion of the large intestine usually shows zebra stripe markings.

8. Diagnosis:

The history, signs and lesions are valuable in reaching a diagnosis. However, because of the similarity of these features to those of other diseases, discussed later, a confirmatory laboratory diagnosis is necessary.

9. Differential Diagnosis:

Diseases that resemble rinderpest clinically are acute mucosal diseases, bovine malignant catarrhal fever, acute coccidiosis, and foot-and-mouth disease. Inoculation of animals with these disease agents usually does not result in mortality. Since some strains of rinderpest are of low virulence, these other diseases may present difficulties in differential diagnosis.

10. Collection of Specimens for Laboratory Confirmation:

For virus isolation, heparinized blood, mesenteric lymph nodes, and spleen are collected early in the acute phase of the disease. One portion of the heparinized blood should be shipped refrigerated; other specimens should be received frozen at the laboratory. Serum should also be collected from animals which have been ill for the longest period of time during the outbreak.

For histopathology, specimens of tonsils, liver, spleen, kidney, and portions of intestines showing lesions should be collected in 15 percent neutral buffered formalin.

11. Laboratory Confirmation:

Attempts to isolate the virus in tissue culture or animals are carried out. Extracts from lymph nodes of infected animals may be used in the complement fixation (CF) or agar gel diffusion precipitation (AGDP) test as antigens against RP rabbit hyperimmune serum. Virus neutralization tests in cell cultures may be carried out with the sera of animals that were sick long enough to develop antibodies. A definitive diagnosis may be obtained by cross-protection tests using immune and susceptible cattle.

12. References:

Dardiri, A. H., Yedloutschnig, R. J., and Taylor, W. D. Clinical and Serological Response of American White-Collared Peccaries . . . . U.S. Animal Health Association Proc. 73: 437-452. 1969.

Plowright, W. Rinderpest Virus. Virol. Monogr. 3: 25-110. (Ed. by S. Gard., C. Hallauer, and K. F. Meyer.) 1968. New York: Springer-Verlag.

2. AFRICAN HORSE SICKNESS

1. Definition:

African horse sickness (AHS) is a highly fatal, insect-borne, febrile, virus disease of Equidae, clinically dominated by an acute pulmonary edema or subacute cardiac form associated with localized areas of inflammatory edema and hemorrhage.

2. Etiology:

The disease is caused by a viscerotropic virus of which nine serologic types have been identified. Mouse-adapted strains are used for vaccines. More recently, murine virus has been propagated in cell cultures which are also useful as a source of relatively inexpensive vaccines for control of outbreaks.

3. Transmission:

The disease is transmitted by arthropods of various Culicoides species. The disease may persist through seasons devoid of insects (including overwintering), as well as in the absence of Equidae. The reservoir host is not yet established.

4. Hosts:

Horses, mules, and donkeys are the natural hosts. Ferrets, mice, and dogs have been infected experimentally; dogs may become transient virus carriers after eating large quantities of infected horsemeat and blood.

5. Clinical Signs:

When newly introduced into a susceptible equine population, the disease may appear in one of three forms: (1) a severe pulmonary disease with a 3- to 5-day incubation period; (2) a subacute or cardiac form associated with swellings of the head, neck, eyelids, cheeks, brisket, thorax, and ventral region of the abdomen (the most characteristic clinical sign is the prominent bulging of the supraorbital fossa); and (3) a subclinical form with a high temperature (104° F) for 1 to 2 days and a short period of general malaise.

6. Gross Lesions:

The most characteristic change is gelatinous edema of subcutaneous and intramuscular tissues, especially in the region of the temple, eyes, and throat. Edema of the lungs is common, as well as endocardial ecchymoses. In some cases there are large quantities of yellowish or sanguinous fluid in the plural cavity and pericardium. Congestion of the fundic portion of the stomach is common.

7. Diagnosis and Differential Diagnosis:

The characteristic seasonal occurrence, history, and clinical signs may assist in reaching a field diagnosis. Signs, such as edema of the supraorbital fossae, subcutaneous edema, edema of the lungs, excess of pleural and pericardial fluid, are further evidence to suspect AHS.

Diseases that may be confused with AHS are anthrax, equine infectious anemia, and equine viral arteritis.

8. Collection of Specimens for Laboratory Confirmation:

Blood for viral isolation should be collected in ethylenediaminetetraacetic acid (EDTA) or potassium oxylate, phenol, and glycerine (OCG) solutions (anticoagulent preservative) at or before the febrile period. Blood is also collected for serum 5 to 6 days following the peak of the temperature rise. At necropsy, a portion of the spleen is collected aseptically and may be placed in glycerol buffer. Other specimens, such as brain, heart, liver, and kidney, should be collected and refrigerated for bacterial examinations.

9. Laboratory Confirmation:

Susceptible Equidae are inoculated intravenously with blood and spleen suspensions; these animals are observed for signs and lesions characteristic of AHS and to obtain virus and sera. The virus may be isolated by intracerebral inoculation of 2- to 6-day-old mice with diluted blood or spleen suspensions. These mice may show nervous signs in 6 to 7 days postinoculation (DPI). However, it is usually necessary to make 2 to 3 passages in mice to obtain high virus concentration in brain tissue.

The brains are harvested from mice in extremis for preparation of complement fixing (CF) antigen, which is used with reference antiserum. The test is group specific for AHS virus types. The CF antibody is at its peak in the sera of infected Equidae 5 to 6 days after the cessation of the febrile period; following this it declines rapidly. The identification of the serotypes of the virus is done by cross-neutralization tests in suckling mice or cell cultures. It is necessary to have available for these tests all nine types of virus and their homologous antisera.

10. Reference:

Howell, P. G. African Horse Sickness. In Emerging Diseases of Animals, FAO Agr. Studies, 61: 71-108. 1963. Rome.

3. AFRICAN SWINE FEVER

1. Definition:

African swine fever (ASF) is a highly contagious, usually acute, viral disease of domestic swine characterized by fever, marked cyanosis of skin areas, and pronounced hemorrhages of the internal organs, particularly the lymph nodes, kidney, and gastrointestinal mucosa. Mortality frequently approaches 100 percent in initial epizootics.

2. Etiology:

The causative agent of ASF is a DNA virus which is 175 to 215 nm in diameter. It is sensitive to lipid solvents and Ortho-phenylphenol disinfectants but is resistant to strong acids and alkalies. The virus causes hemadsorption of swine red cells in infected leukocyte cultures. Inclusion bodies are found within the cytoplasm cells infected with the virus.

It remains viable at refrigeration temperatures for 18 months. In 1957 the disease appeared in Portugal, presumably imported by accident from Africa. From there the disease spread to Spain. By 1967 it was reported in Italy; and in 1971 it was found in Cuba, posing a serious threat to the swine population in the Western Hemisphere.

3. Transmission:

Infection is most common as a result of contact with recovered or carrier pigs and ingestion of contaminated or infected garbage, urine, feces, and carcasses. Recently, transmission was achieved in Africa and Spain with infected ticks.

4. Hosts:

Pigs, wart hogs, forest hogs, and bush pigs are proved reservoirs. The American javelina is resistant.

5. Clinical Signs:

In acute and subacute forms, the incubation period is 5 to 15 days. Fever, depression, lachrymal discharge, cough, diarrhea, dehydration, and death are typical signs; the course of the acute disease is 6 to 12 days.

6. Gross Lesions:

Lesions closely resemble those of hog cholera (HC), except that they may be more severe. Hemorrhages are found on the epicardium and endocardium. Lymph nodes are hemorrhagic. Spleen enlargement and splenic infarcts are common. Petechial hemorrhages of the kidneys and urinary bladder occasionally are found.

As ASF became enzootic in Spain and Portugal, the signs and lesions in a large proportion of animals lessened in severity and were more similar to those of HC than they had been previously.

7. Diagnosis:

Disease signs and lesions may or may not be suggestive of ASF. Marked severity of lesions, especially in pigs which were previously vaccinated for HC, may lead to a presumptive diagnosis.

8. Differential Diagnosis:

The disease should be differentiated from HC, erysipelas, and salmonellosis. Appropriate specimens must be taken for all suspect diseases.

9. Collection of Specimens for Laboratory Confirmation:

For virus isolation, spleen, gastrohepatic, and mesenteric lymph nodes are the organs of choice since they contain high virus concentrations. They are shipped to the laboratory preferably on dry ice. Where chronic ASF is suspected, serum should be obtained from swine infected longest and shipped frozen.

10. Laboratory Diagnosis:

African Swine Fever can be diagnosed in the laboratory by (1) inoculation of suspect material into immune and susceptible pigs; (2) demonstration of the hemadsorption reaction in pig buffy coat cultures inoculated with blood or spleen suspensions from the suspect pig; and (3) paired sera from the suspect animals which lived the longest may be tested by the AGDP or the immunoelectroosmophoresis (IEOP) tests. However, these serologic tests necessitate the use of specially prepared cell culture antigens and known ASF immune serum. The fluorescent antibody (FA) technique for testing liver, spleen, and other frozen tissue sections and smears is also a reliable method for detection of ASF.

Recent comparative research on ASF and HC by the Commission of the European Communities (Rabot, 1971) reports the following: "Non-purulent panencephalitis. . .involving both the grey and the white matter is a very important characteristic of HC." This was found in 72 percent and satelitosis in 60 to 100 percent of the HC cases investigated. On the other hand, brain damage in ASF was characterized by cell degeneration, ranging from acute swelling and simple retraction to the severe cellular disease of Nissl; perivascular mononuclear infiltration was generally "not very severe."

11. References:

DeTray, D. E. African Swine Fever. Adv. Vet. Sci. 8: 299-313. 1963.

Hess, W. R. African Swine Fever Virus. Virology Monog. 9: 1-33. 1971. Springer-Verlag.

Rabot, L. B. Properties of the Virus of Classical Swine Fever. European Community, Off. Offic. Pub., Pub. 8. 1971.

4. FOWL PLAGUE

1. Definition:

Fowl plague (FP) is an acute, highly contagious, fatal viral disease of chickens and turkeys. Other birds such as waterfowl, sparrows, pheasants are also affected.

2. Etiology:

Fowl plague is a myxovirus that can remain viable for long periods of time in infected tissues. It causes hemagglutination of the erythrocytes of chickens.

3. Transmission:

Direct contact with aerosols from infected birds is the main method of transmission. The disease is also spread by contaminated feed and equipment.

4. Hosts:

The chief hosts are chickens and turkeys, although other avian species are susceptible.

5. Clinical Signs:

Depression, drooping of feathers and tail, loss of appetite, cyanosis, and swelling of the comb and wattles are common signs.

6. Gross Lesions:

Hemorrhages in various parts of the body are common; these are more striking in the submucosal tissues of the proventriculous. Petechiae are found on the heart, serous intestinal surfaces, and the peritoneum. Hemorrhage in the mucous membranes lining the gizzard is also common.

7. Diagnosis and Differential Diagnosis:

Severe mortality in susceptible chickens accompanied by the signs and lesions described previously lead to a presumptive diagnosis. The syndrome must be distinguished from virulent Newcastle disease and fowl cholera.

8. Collection of Specimens for Laboratory Confirmation:

Specimens should be collected from several birds. Trachea, spleen, lungs, liver, and blood are the tissues of choice. These should be frozen and transmitted on dry ice.

9. Laboratory Diagnosis:

Virus isolation is achieved by inoculation of 9-day chicken embryos. At death, the allantoic fluid is harvested and tested for hemagglutination of chicken erythrocytes. The isolated agent is then identified by hemagglutination inhibition and virus neutralization tests using specific hyperimmune serum.

10. References:

Easterday, B. C., and Tumova, B. Avian Influenza. In Diseases of Poultry, M. S. Hofstad, ed., pp. 670-700. 1972. Iowa State Univ. Press, Ames.

Stubbs, E. C. Fowl Plague. In Diseases of Poultry, H. E. Biester and L. H. Schwartz, eds., 5th ed., pp. 813-822. Iowa State Univ. Press, Ames.

5. CONTAGIOUS BOVINE PLEUROPNEUMONIA

1. Definition:

Contagious bovine pleuropneumonia (CBPP) is a specific disease of cattle caused by Mycoplasma mycoides, subspecies mycoides. It is highly infectious and occurs in acute, subacute, and chronic septicemic forms.

2. Etiology:

Mycoplasma mycoides mycoides is a pleomorphic organism that is sensitive to drying and disinfectants. The causative organisms of contagious pleuropneumonia of goats and sheep share similar cultural and antigenic features with CBPP but are species specific.

3. Transmission:

The organism is transmitted through inhalation of dried bronchial secretions from infected carrier animals.

4. Hosts:

Cattle of all ages may be infected.

5. Clinical Signs:

The incubation period is usually from 3 to 6 months long but it may be shorter: in highly susceptible cattle the natural disease has been known to develop in from 10 to 14 days. Initial signs are fever, cessation of rumination, and severe cough after exercise. Other signs are an arched back, chest pain, distended elbows, and extended head and neck. Grunting expiration, shallow rapid breathing with fluid sounds occur, then gurgling rales, pleuritic friction, and areas of dullness on percussion follow.

6. Gross Lesions:

Typical lesions found at necropsy are:

Thickening and inflammation of the pleura with occurrence of fibrin deposits. Interlobular edema in one or both lungs. The "marbled" appearance of classical descriptions is caused by distension of the interlobular septa and is accompanied by areas of gray to red hepatization. In the chronic forms of the disease necrotic areas may be walled off by connective tissue capsules forming characteristic sequestra, which may persist for a long time.

7. Diagnosis:

Contagious bovine pleuropneumonia is suspected when the marbled appearance of lobules and the presence of a large quantity of straw-colored fluid in the thoracic cavity are found at necropsy.

8. Differential Diagnosis:

The lungs of animals which die of East Coast Fever may have a similar appearance to those which have CBPP. Subacute pasteurellosis sometimes may be confused with CBPP.

9. Collection of Specimens for Laboratory Confirmation:

Samples from lung lesions, pleural fluids, lymph nodes, and lung tissue exudate are collected and frozen for isolation of the organisms. Samples from lung, spleen, brain, liver, and kidney are preserved in formalin for histopathologic examination. If possible, acute and convalescent sera are obtained.

10. Laboratory Diagnosis:

Various serologic tests are used, including CF and agglutination. Metabolic and growth inhibition tests to identify the specific mycoplasma organism have been used successfully. The FA test is also used.

11. References:

Henning, M. W. Pleuropneumonia Contagiosa Bovium, Lung Sickness of Cattle, Longsiekte. In Animal Diseases of South Africa, 3d ed., pp. 204-229. 1956. Central News Agency Ltd., Johannesburg.

Hudson, J. R. Contagious Bovine Pleuropneumonia. FAO Agr. Studies, No. 86. 1971. Rome.

6. BOVINE HERPES DERMOPATHIC DISEASE

1. Etiology:

Bovine herpes dermopathic (BHD) disease is caused by herpes viruses that are similar in their biological, immunological, and physicochemical characteristics. Intranuclear inclusions, multinucleated, and giant cells develop in the skin of infected animals and cell cultures.

2. Transmission:

The exact mode of transmission is not known; however, biting insects and milking methods are suspected of spreading the disease.

3. Hosts:

Cattle and buffaloes of all ages are susceptible.

4. Clinical Signs and Gross Lesions:

The incubation period is 1 to 2 weeks. A fever of several days duration precedes the formation of cutaneous nodules. The nodules are first round; later they flatten and become exudative and are covered by dry scabs. When the scabs fall off, the hairless skin is normal. Bovine mammillitis lesions are chiefly restricted to the teats and udder skin and tend to become ulcerative. A large proportion of cattle herds in enzootic areas develop neutralizing antibodies without having noticeable disease signs or lesions.

5. Differential Diagnosis:

Signs and lesions are indistinguishable from those of lumpy skin disease and skin infections caused by Dermatophilus congolensis, pox, and pseudopox viruses. Lesions in the epithelium of the oral and nasal cavities cause excessive salivation, and the signs may be confused with mucosal and vesicular diseases.

6. Collection of Specimens for Confirmatory Diagnosis:

Skin lesions may contain the virus when they are fresh and exudative. A viremia is present for approximately 4 days after appearance of skin lesions. Virus may also be obtained from vesicular fluids and from exudative teat, ear, and tail lesions. Blood samples should be taken from several affected animals in early and late disease stages to obtain paired sera. The specimens should be frozen with dry ice and sent to the laboratory.

7. Laboratory Diagnosis:

To isolate virus, fluids from lesions and skin triturates from lesions are inoculated into primary bovine kidney cell cultures. The infected tissues and cultures may be examined by the electron microscope to demonstrate herpes virus morphology. Cultures stained with hemotoxylan and eosin (H and E) permit demonstration of intranuclear inclusion bodies and syncytial cytopathogenicity. The isolated virus is identified by using reference serum from convalescent animals to conduct virus neutralization and fluorescent antibody (FA) tests. Susceptible cattle, so determined by negative serologic tests for antibodies, can be inoculated intravenously to reproduce the disease and to obtain optimal tissue and blood samples.

8. References:

Martin, W. B., Martin, B., Lander, I. M., and Pirie, H. M. Bovine Mammillitic Virus Infection (BVM) and Lumpy Skin Disease. Vet. Rec. 86: 661-662. 1970.

Breese, S. S. Jr., and Dardiri, A. H. Electron Microscopic Characterization of a Bovine Herpes Virus from Minnesota. J. Gen. Virol. 15: 69-72. 1972.

7. DUCK PLAGUE
(DUCK VIRUS ENTERITIS)

1. Definition:

Duck plague (DP) or duck virus enteritis (DVE) is a disease of domestic ducks, geese, and wild waterfowl characterized by bodily hemorrhage and pathognomonic esophageal and cloacal lesions.

2. Etiology:

The disease is caused by a herpes virus approximately 180 nm in diameter with a core of 75 nm. The virus was attenuated by passage in chicken embryos and cell cultures of the same origin. The virus produces plaques in monolayer cultures. There is evidence of variation in virulence of different isolates, but it appears that all are immunologically similar.

3. Transmission:

The virus is transmitted by contact of susceptible birds with infected ones as well as both direct and indirect exposure to contaminated material and equipment.

4. Hosts:

Only birds belonging to the family Anatidae, such as ducks, geese, and swans, are known to harbor the virus.

5. Clinical Signs:

The incubation period is 3 to 7 days, and all ages of birds are susceptible. Egg production may drop 25 to 40 percent in a flock which is affected. Ducks are unable to stand, their eyes are congested, and the feathers around the eyes are matted. When attempting to move, ducks creep with their wings outstretched. Young ducks, 2 to 7 weeks of age, show signs of dehydration and have diarrhea. Mortality may be 25 to 100 percent. A subclinical form of the disease may prevail in some flocks.

6. Gross Lesions:

In mature and young ducks tiny hemorrhagic spots corresponding to the lymphoid areas located in the esophageal mucosa appear in longitudinal rows. Later in the disease the esophageal mucosa is covered with a yellow or gray irregular pseudomembrane. Similar lesions may appear on the cloacal mucosa. In young ducklings dark-reddish, macular bands appear in the mucosal surfaces of the small intestines. Extravasation of blood in abdominal and chest cavities as well as petechiation of the heart are associated with the disease. Secondary bacterial infections are common. The lesions in the esophagus are considered pathognomonic.

7. Diagnosis:

When present, the lesions in the esophagus may be considered adequate evidence for a diagnosis.

8. Differential Diagnosis:

This requires consideration of the possibility of exposure of a flock to other diseases such as duck virus hepatitis, fowl plague, velogenic Newcastle disease, and pasteurellosis.

9. Collection of Specimens and Laboratory Confirmation:

Duplicate portions of esophagus, intestines, liver, and cloacal tissues with lesions are collected; one portion frozen for virus isolation and the other in a fixative for demonstration of Type A intranuclear inclusion bodies. Liver and spleen are macerated for virus isolation; the triturates are inoculated into 9- to 11-day-old duck embryos via the chorio-allantoic membranes. An isolate that is lethal to duck embryos with extensive bodily hemorrhage is suggestive of DVE. The neutralization of such an isolate by known DVE antiserum confirms the identification. An increase in serum neutralization titer of 1.75 ($\log_{10}$) in the serums of convalescent waterfowl is indication of recent infection.

10. References:

Dardiri, A. H. Attenuation of Duck Plague Virus and Its Propagation in Cell Culture. Archiv. fur gesamte Virusforschung 27: 55-64. 1969.

Leibovitz, Louis. Gross and Histopathologic Changes of Duck Plague (Duck Virus Enteritis). Amer. J. Vet. Res. 32: 275-290. 1971.

Leibovitz, Louis. Diseases of Poultry. 6th ed. pp. 732-744. 1972. Iowa State University Press, Ames.

8. TESCHEN DISEASE OF PIGS

1. Definition:

Teschen disease (TD) is a virus disease of swine characterized by incoordination, convulsions, spasms, and paralysis.

2. Etiology:

Strains of these viruses vary greatly in virulence. The Tyrol subtype 2 virus is highly virulent while the Talfan virus in England produces either a mild syndrome or an inapparent infection. All of the viral strains are immunologically similar. The virus is resistant to drying and infective only for pigs. It is destroyed by 3 percent NaOH in less than 3 hours.

3. Transmission:

Transmission is essentially by the oral route although experimental intranasal infection is successful.

4. Hosts:

Domestic swine are the only known hosts.

5. Clinical Signs:

The major clinical signs of the virulent disease are those of encephalomyelitis. They include a thermal response (104° to 106°o F), loss of appetite, hyperesthesia, muscular rigidity, convulsions, immobility, clonic spasms, paralysis of hind legs, recumbance, and death.

6. Gross Lesions:

No gross lesions are found in the nervous system; but microscopic lesions characteristic of aseptic encephalomyelitis are found, especially in the gray matter of the central nervous system. These are concentrated chiefly in the spinal cord and cerebellum and consist of perivascular cuffing and degeneration of the neurons.

7. Diagnosis:

Diagnosis depends upon the case or outbreak history, histopathology, transmission of the disease to colostrum-deprived pigs, virus isolation, and viral neutralization tests.

8. Differential Diagnosis:

Teschen disease should be differentiated from the nervous reactions of swine which are associated with other enteroviruses, hog cholera, swine erysipelas, prolonged periods of nervousness associated with vitamin A deficiencies, and rickets caused by pantothenic acid deficiencies.

9. Collection of Specimens and Laboratory Diagnosis:

Portions of the brain, cerebellum, medulla, and spinal cord should be collected in 15 percent buffered formalin for histopathologic study. Samples of the same material should be obtained aseptically for virus isolation and the viral neutralization test.

10. References:

Dardiri, A. H., Seibold, H. R., and Delay, P. D. The Response of Colostrum-Deprived, Specific Pathogen-Free Pigs to Experimental Infection with Teschen Disease Virus. Can. J. Comp. Med. Vet. Sci. 30 (3): 71-81. 1966.

Grig, A. S., Bannister, G. L., Mitchell, D., and Corner, A. H. Studies on Pathogenic Enteroviruses. II. Isolation of Virus in Tissue Culture, Brain and Feces of Clinical Cases. Can. J. Comp. Med. Vet. Sci. 25: 142-160. 1961.

9. FOOT-AND-MOUTH DISEASE

1. Definition:

Foot-and-mouth disease (FMD) is an acute, highly communicable disease existing almost exclusively in cloven-footed animals, domesticated and wild. The disease is characterized by the formation of vesicles and erosions in the mucosa of the mouth and external nares (especially on the snout of pigs) and the skin between and above the hooves of the feet; other areas, including mammary tissue, may be involved.

2. Etiology:

The disease is caused by a virus first isolated in 1897; it is classified with the enteroviruses as a member of the picornavirus group. It has a single-stranded ribonucleic acid core with a protein coat which appears to consist of 32 capsomeres forming a symmetrical icosahedral capsid with a diameter of about 23 nm. There are 7 immunologically and serologically distinct types of virus identified as Types O, A, and C; Southern African Territories (SAT-1, SAT-2, SAT-3), and Asia-1. Within the 7 types at least 61 subtypes have been designated by CF tests.

3. Geographical Distribution:

FMD occurs in most of the major livestock producing countries of the world, except North America, Central America, Australia, New Zealand, Japan, and Ireland. Several countries in Europe, especially Great Britain and some of the Scandinavian countries, are generally free for periods of several years; for example, FMD has not occurred in Great Britain during the last 4 years.

Type Distribution:

Types O, A, and C occur in various parts of the world while the African types, SAT-1, SAT-2, and SAT-3, were not found outside Africa until 1962 when an epizootic due to SAT-1 occurred in the Middle East. Asia-1 has been identified from Pakistan, India, Israel, Iran, Iraq, Hong Kong, Thailand, and other Near and Far Eastern countries.

4. Transmission:

The virus is transmitted by contact with infected animals (aerosols primarily), by infected animal products, and by contaminated objects.

5. Hosts:

All cloven-hooved animals, domestic and wild, are naturally susceptible; pathogenesis for some species is reduced in certain strains. The hedgehog, muskrat, armadillo, and perhaps other wild animals besides the cloven-hooved ones are susceptible; in addition, a wide variety of laboratory animals and cell culture systems can be infected with FMD virus. Man is rarely infected but is capable of transmitting the virus.

6. Clinical Signs:

In cattle, characteristic signs are a moderate pyrexia, lassitude, anorexia, excessive salivation, smacking of the lips, and drooling; these accompany the formation, rupture, and erosion of vesicles of the mouth. When the feet are involved, lameness is seen. Reduced lactation, mastitis, and abortions are common. Mortality in young animals may be as high as 50 percent but is seldom above 5 percent in adults. Swine show many similar signs; lameness with a changed gait may be quite evident. The incubation period is from 1 to 5 days or longer.

7. Gross Lesions:

Vesicles are not pathognomonic for FMD alone, since they are also associated with vesicular stomatitis (VS), vesicular exanthema of swine (VES), and swine vesicular disease (SVD). Classical vesicular lesions may not be found; when they occur they usually rupture leaving eroded, hemorrhagic, granular mucosal surfaces of the nose and mouth, as well as the skin, epithelial tissues of the feet and other regions.

Gastrointestinal lesions may be found at necropsy, particularly of the rumen. In rare cases lesions of the perineum, vulva, or scrotum are seen. Tiger heart (gray, white, or yellowish myocardial lesions) may be seen in calves. In swine and sheep, lesions on the tongue are usually smaller than those of cattle.

8. Diagnosis:

Diagnosis by clinical signs is virtually impossible.

9. Differential Diagnosis:

The inoculation of susceptible horses, swine, and cattle (brought from a region distant from the outbreak) with suspect material may be helpful in differentiating one vesicular disease from another. All three of the species are susceptible to VS; cattle and swine are susceptible to FMD; swine alone respond to SVD and VES. However, laboratory confirmation is necessary.

10. Collection of Specimens for the Laboratory:

Specimens include the following:

Esophageal-pharyngeal fluid obtained with a probang deposited in sterile tissue culture medium containing antibiotic; vesicle fluids collected with aseptic technique in a sterile vial; lesion scrapings placed in tissue culture medium containing antibiotic; paired sera from individual animals or sera from separate animals taken at early and later stages. All specimens are immediately frozen for shipment (preferably) or placed in glycerol.

11. Laboratory Confirmation:

Laboratory tests for confirmation include:

Complement fixation, the AGDP, virus neutralization, and cross-immunity tests.

12. Control:

In countries where the disease is endemic, incidence of the disease is controlled by vaccination programs. In an increasing number of countries vaccination is mandatory; in others it is voluntary. In countries that are generally free of the infection, the disease is eradicated by slaughter followed by disinfection of the premises. The animal carcasses are generally destroyed by burning or burial. While costly, this method is considered to be the most effective way to deal with an outbreak.

13. References:

Bachrach, H. L. Foot-and-Mouth Disease. Annu. Rev. Microb. 22: 201-244. 1968.

Callis, J. J., Shahan, M. S., and McKercher, P. D. Foot-and-Mouth Disease. Diseases of Swine. 3d. ed., H. W. Dunne, ed., pp. 309-336. Iowa State Univ. Press, Ames.

Cottral, G. E. Diagnosis of Bovine Vesicular Diseases. J. Amer. Vet. Med. Assoc. 161 (11): 1293-1298. 1972.

10. PESTE DES PETITS RUMINANTS

1. Definition:

Peste des petits ruminants (PPR) is an acute, subacute, or chronic disease of goats and sheep.

2. Etiology:

The disease is caused by a virus physically and chemically similar to that of rinderpest. PPR virus also is serologically and immunologically related to the measles and distemper, as well as to rinderpest viruses.

3. Transmission:

Transmission occurs readily between sick and healthy goats and sheep, with the latter showing signs. The virus is also transmissible to cattle that are susceptible but without development of signs or lesions.

4. Hosts:

Sheep and goats are apparently the natural hosts, with the latter more susceptible.

5. Clinical Signs:

The incubation period is from 4 to 10 days. A febrile reaction is followed by watery nasal discharge which becomes mucopuralent; encrustation of the external nares accompanies drying of the discharge. The fever is followed by the appearance of mucosal lesions and by diarrhea. Females develop labial erosions. Frequently, affected animals develop respiratory signs.

6. Gross Lesions:

Lesions are found in the lungs and alimentary tract. Extensive erosive stomatitis develops in the oral mucosa accompanied by hemorrhagic gastroenteritis. The Peyer's patches are congested and necrotic; "zebra stripe" markings are prominent. Bronchopneumonia is common.

7. Diagnosis:

A presumptive diagnosis is made when an epizootic characterized by the signs and lesions enumerated above appears in sheep or goats and is accompanied by high mortality.

8. Differential Diagnosis:

Differentiation from bovine rinderpest, bluetongue, pox, and contagious pustular dermatitis is necessary.

9. Collection of Specimens and Laboratory Confirmation:

Blood, spleen, and mesenteric lymph nodes from sick animals and those in extremis are specimens of choice for submission to the laboratory. Serums from recovered animals are necessary for detection of antibodies. Cross-protection tests with susceptible goats and cattle are carried out with PPR and RP. Virus neutralization tests may be conducted in cell cultures.

10. References:

Mornet, P., Orue, J., Gilbert, Y., Theiry, G. et Sow Mamdou. La peste des petits ruminants en Afrique Occidentale Francaise. Ses rapports avec La peste bovine. Rev. Elev. Med. Vet. Pays Trop. 9: 313-342. 1956.

Rowland, A. C., and Bourdin, P. The Histological Relationship Between "Peste des Petits Ruminants" and Kata in West Africa. Rev. Elev. Med. Vet. Pays Trop. 23: 301-307. 1970.

11. LUMPY SKIN DISEASE[1/]

1. Definition:

Lumpy skin disease (LSD) in classical (Neethling) form is an acute virus disease of cattle, characterized by the eruption of variably sized cutaneous nodules, edema of one or more limbs, and swelling of the superficial lymphatic glands.

2. Etiology:

The disease is caused by a pox virus (Neethling) that is related serologically to sheep and goat pox viruses.

3. Transmission:

Insect transmission is considered more important than is contact transmission.

4. Hosts:

Cattle and buffaloes are the natural hosts of the virus. It has been isolated and propagated in lamb testes and lamb kidney cell cultures.

5. Clinical Signs:

The incubation period is 4 to 14 days. There is a fluctuating fever, increased salivation, and nasal discharge. Skin eruptions occur following the peak of temperature rise. Skin nodules appear in different parts of the skin. They are easily seen on the neck, back, thighs, perineum, vulva, and around the muzzle. Lesions on the muzzle, the ventral surface of the tail, and ears are yellowish in color, covered with brownish lumph exudate and surrounded by an intensely congested zone. Mild lesions heal in a few weeks.

1/ Lumpy Disease, Pseudourticaria, Knopvelsiekte.

6. Gross Lesions:

The skin nodules vary in size; they are thickened masses of skin tissue of a creamy gray color, sometimes containing caseous material. In mild cases the skin lesions are round, circular, and involve the superficial skin layers. Ulcerated lesions may be found in the mucous membrane of the mouth, nose, and larynx. Similar lesions may be found on the vulva.

7. Diagnosis and Differential Diagnosis:

The nodular eruptions of the skin and mucous membranes, swelling of the limbs, and lymphatic glands are useful in making a presumptive diagnosis.

The disease must be differentiated from the Allerton type and related bovine herpes virus infections that also cause skin lesions. Other disease conditions that may be confused with LSD are allergies, screwworm infestations, and cutaneous streptothricosis.

8. Collection of Specimens for Laboratory Confirmation:

Fresh skin lesions should be harvested, and specimens of swollen lymph glands should also be collected and preserved on dry ice. Duplicate tissue samples are also preserved in formalin for histological examination. Both acute and convalescent sera from several animals should be obtained and frozen.

9. Laboratory Diagnosis:

Cytopathogenicity and cytoplasmic inclusion bodies in cell cultures may be found. Inhibition of both features may be accomplished by known antiserum. Electron micrographic examination of skin tissues or cell monolayers may reveal the causative agent pox virus. Fluorescent antibody and ferritin tagging techniques are useful in identification of the virus particles in infected skin specimens and cell cultures.

10. Reference:

Weiss, K. E. Lumpy Skin Disease. In Emerging Diseases of Animals, FAO Agr. Studies 61: 179. 1963. Rome.

12. EPHEMERAL FEVER

1. Definition:

Ephemeral fever (EF) is an acute, febrile disease of cattle characterized by shivering, muscular stiffness, and, occasionally, enlargement of the peripheral lymph nodes.

2. Etiology:

The disease is caused by an insect-borne virus. Blood stored at +2° C to -2° C remains infectious for about 6 weeks, but lyophilized buffy coat fractions stored at -70° C retain infectivity for at least 3 years. The virus has been adapted to infant mice and cell culture.

3. Geographic Distribution:

The disease is enzootic or has been recognized in Africa, India, Japan, the East Indies, and Australia.

4. Transmission:

The disease agent is known to be transmitted by arthropods; sand flies are regarded as the chief vector.

5. Hosts:

Bovidae are the only known hosts.

6. Clinical Signs:

The disease usually lasts about 3 days. The animal exhibits clinical signs of fever, followed by stiffness of the musculature for 1 or 2 days to a week, followed by recovery. Abortion may occur in cows during late pregnancy, and there may be a temporary interference with lactation. Mortality is 0.5 percent or less; stress from hot weather and foot travel may increase morbidity and mortality.

7. Gross Lesions:

At necropsy, no visible lesions have been reported in uncomplicated cases.

8. Diagnosis:

The disease may be diagnosed easily when it occurs in epizootic form. However, sporadic cases are easily confused with other diseases.

9. Differential Diagnosis:

The disease should be differentiated from mild Rift Valley fever and other febrile diseases in their early stages.

10. Collection of Specimens for Laboratory Confirmation:

Hyperemic defibrinated blood should be sent refrigerated; early and convalescent sera may be submitted frozen. Laboratory diagnosis is made by transmission of the disease to susceptible cattle or by the CF test.

To transmit the disease, the buffy coat fraction of blood from sick animals is the material of choice.

11. Reference:

Heuschele, W. F., and Johnson, D. C. Response of Cattle to Attenuated and Virulent Virus. II. U.S. Animal Health Assoc. Proc. 73: 185-195. 1969.